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EDITORIALS

Dietary therapy for irritable bowel syndrome

High expectations for low FODMAP diets

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Irritable bowel syndrome (IBS) is a common disorder of the digestive system, affecting about 10% of the global population. The condition has no definitive biomarker or cure, but various drug treatments have been introduced in recent years, including antibiotics (to treat presumed small intestinal bacterial overgrowth) and agents that affect motility through fluid secretion or the enteric nervous system. Despite these advances, perhaps the most popular option among patients in recent years has been a dietary approach, the "low FODMAP diet."

The term FODMAP was first coined by Gibson and Shepherd in 2005, referring to a new dietary class comprising fermentable oligosaccharides, disaccharides, monosaccharides, and polyols. The authors identified a list of foods that are highly fermented but poorly absorbed, leading to the expansion of ileocolonic bacteria. This broad dietary class includes fructose (in fruits and sweeteners), lactose (in dairy products), fructans (wheat based products), galacto-oligosaccharides (legumes), and polyols such as xylitol and mannitol (fruits and artificial sweeteners). The authors first proposed FODMAPs as a potential trigger for inflammatory bowel disease (Crohn's disease and ulcerative colitis) because of their effect on the ileocolonic microbiome. Although the low FODMAP diet has become an increasingly studied strategy for treating inflammatory bowel disease, data are limited to small pilot studies.6

A dietary approach to treating irritable bowel syndrome has intuitive appeal. Most patients with this condition report that their symptoms are exacerbated by specific foods or are temporally correlated with eating in general. There is also a pathophysiological rationale, given the effect of these foods on the production of gas and short chain fatty acids, and the effect of the fatty acids on motility. Indeed, evidence is accumulating that this strategy is effective. A randomised crossover trial of 30 patients in Australia with irritable bowel syndrome (including those with diarrhoea predominance, constipation predominance, and a mixed pattern of symptoms) found that those prescribed a low FODMAP diet had lower (improved) gastrointestinal symptom scores on a visual analogue scale compared with those prescribed a typical Australian diet (22.8 versus 44.9, P<0.001). A subsequent systematic review and meta-analysis identified six randomised trials of the low FODMAP diet, with a pooled analysis finding a significant decrease in symptom severity scores (odds ratio 0.44; 95% confidence interval 0.25 to 0.76).

The rise of the low FODMAP diet for irritable bowel syndrome has had a ripple effect on another condition, non-coeliac gluten sensitivity. People with this syndrome, which has uncertain pathogenesis and natural course, report both intestinal (eg, bloating, abdominal pain, and altered bowel habits) and other symptoms (eg, fatigue and headache) when exposed to dietary gluten but have had coeliac disease definitively ruled out. A randomised trial examining the syndrome found that affected patients had a greater exacerbation of symptoms when given gluten than when given placebo, providing initial justification that non-coeliac gluten sensitivity is a distinct clinical entity. However, in a second trial, when patients with this syndrome were put on a low FODMAP diet before randomisation, introduction of gluten made no difference to symptoms relative to placebo. The investigators concluded that the apparent improvement reported by patients who start a gluten-free diet may be due to restricting FODMAPs. Thus a potential breakthrough in the treatment of irritable bowel syndrome has caused those studying non-coeliac gluten sensitivity to rethink their assumptions.

The fact that the low FODMAP diet seems effective is welcome news for patients with irritable bowel syndrome and their doctors. However, some caution is warranted. The long term effectiveness of this diet is unknown. In the six randomised trials included in the recent meta-analysis, the longest duration of dietary intervention was six weeks. Given the restrictive nature of the diet, it may be hard to stick to. Even among patients who can adhere to the diet in the long term, it is not known whether it promotes an ileocolonic microbiome conducive to long term health and symptom control. Although one study showed that a three week, low FODMAP diet resulted in greater microbial diversity and reduced total bacterial abundance, longer term data are not available. There is no evidence that the low FODMAP diet is harmful, although inadequate vitamin and mineral intake is a theoretical concern given its overlap with the gluten-free diet.

Given the sheer number of restricted items in a low FODMAP diet, we should consider this diet a potent combination of dietary

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prescriptions. The full spectrum of FODMAPs may not be responsible for symptoms in a given patient, or even in most patients. Future studies should consider breaking down this list of restrictions into component parts to determine whether a less restrictive dietary approach could be effective. A positive result on fructose breath testing might allow the restriction of fructose rather than a full low FODMAP diet. In addition, a positive result may predict response to the FODMAP diet. An experienced dietitian can help manage patients on a low FODMAP diet and facilitate controlled reintroduction of individual components. Ultimately, irritable bowel syndrome may prove to be a heterogeneous group of conditions that respond to a range of dietary strategies. It is likely that one size does not fit all.

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